DETAILED ACTION

Claim amendments filed on 03/11/2010 have been entered.

Claims 7-9, 11, 13, 14, 16-18, and 20 are cancelled. Claims 1-6, 10, 12, 15, 19, and 21 are pending. Claims 1-6, 10, and 12 are amended.

Claims 15 and 19 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim.

Claims 1-6, 10, 12, and 21 are currently under examination.

This application 10/592,010 is a 371 of PCT/EP05/03888 filed on 04/13/2005. Foreign application European Patent Office (EPO) 04008881.7 was filed on 04/14/2004.

Priority

The following statements have been documented in the office action mailed on 11/12/2009.

This application 11/592,010 filed on 03/06/2007, the filed Application data sheet filed on 03/06/2007 claims benefit of foreign application European Patent Office (EPO) 04008881.7 filed on 04/14/2004. The Examiner acknowledges that Applicant has submitted on 09/07/2006 a certified copy of European Patent Office (EPO) 04008881.7 filed on 04/14/2004 under requirement of 35 U.S.C. 119 (a-d) conditions. However, it is noted that, the European Patent Office (EPO) 04008881.7 filed on 04/14/2004 is not in English. Therefore, without a certified translation of European Patent Office (EPO) 04008881.7, the effective filing date for the instant claims is 04/13/2005, which is the filing date of PCT/EP05/03888. Applicant cannot rely upon the foreign priority papers to overcome the rejection under 35 USC 102 (e) or 102 (a) as set forth below because a translation of said papers has not been made of record in accordance with 37 CFR 1.55. See MPEP § 201.15.

In the reply filed by Applicants on 03/11/2010, Applicants provides a translation of EP 0400888.7 and a statement that the translation is accurate. Applicant states that the presently claimed invention finds support throughout the EP 0400888.7 application (see, for example, page 3, lines 9-20 and page 14, line 10, to page 17, line 25, of the translation).

In response, in light of submitted English translation of EP 0400888.7 application, the priority of instant application is determined to be 04/14/2004, the filing date of foreign application EP 0400888.7. For the clarity of record, the Examiner notes that Applicant benefits priority one year before US filing date (filing date of PCT), but not one year before the priority of foreign documents. In other words, a reference published before 04/13/2004 is considered as 102(b) art because it is published one year before 04/13/2005 (filing date of PCT/EP05/03888).

Claim Objection

1. Previous objection of claims 1-6, 10, and 12 for missing an article (i.e. "a" or "an" or "the") in the beginning of these claims, is ***withdrawn*** because the claims have been amended.

Claim Rejection - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless -

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this

subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

2. Previous rejection of claim 1, 2, 4-6, 10, and 12 are rejected under 35 U.S.C. 102(a) and under 35 U.S.C. 102(e) as being anticipated by **Zheng et al.** (US Publication No. 2006/0057102, publication date 03/16/2006, filed on 08/11/2005, provisional applications filed on 08/11/2004), is ***withdrawn***.

In the reply filed by Applicants on 03/11/2010, Applicants provide a translation of EP 0400888.7 and a statement that the translation is accurate. Applicant states that the presently claimed invention finds support throughout the EP 0400888.7 application (see, for example, page 3, lines 9-20 and page 14, line 10, to page 17, line 25, of the translation). Applicants submit that, in view of the submission of the translation of EP 0400888.7, the presently claimed invention is entitled to the benefit of the April 14, 2004 filing date of EP 0400888.7 and Zheng is not available as prior art under 35 U.S.C. § 102(a) or § 102(e).

Applicant's arguments have been fully considered and found persuasive. The rejection is thereby withdrawn.

Claim Rejection - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

3. Previous rejection of claims 1, 4, 6, 10, and 12 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Ferrari-Lacraz et al.** (Ferrari-Lacraz et al. An antagonist IL-15/Fc protein prevents costimulation blockade-resistant rejection, *J. Immunol.* 167(6):3478-85, 2001) in view of **Sutherland et al.** (Sutherland et al., Protective effect of CTLA4Ig secreted by transgenic fetal pancreas allografts, *Transplantation*, 69(9):1806-12, 2000; This reference is cited in the IDS filed on 11/13/2006), is ***withdrawn*** because the claims have been amended.

Amended claim 1 filed on 03/11/2010 reads as follows: An expression system for use in a producer cell line, containing one or more nucleic acid(s) comprising a) at least one nucleic acid for an IL-15/Fc fusion protein, b) at least one promotor and c) at least one nucleic acid for a CD5 leader, the promotor and the nucleic acid for the CD5 leader being functionally linked to the nucleic acid for the IL-15/Fc fusion protein.

Neither Ferrari-Lacraz et al. nor Sutherland et al. explicitly disclosed the amended limitation "for use in a producer cell line".

4. Previous rejection of claims 2, 3, 5, and 21 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Ferrari-Lacraz et al.** (Ferrari-Lacraz et al. An antagonist IL-15/Fc protein prevents costimulation blockade-resistant rejection, *J. Immunol.* 167(6):3478-85, 2001) in view of **Sutherland et al.** (Sutherland et al., Protective effect of CTLA4Ig secreted by transgenic fetal pancreas allografts, *Transplantation*, 69(9):1806-12, 2000; this reference is cited in the IDS filed on 11/13/2006) as applied to claims 1, 4, 6, 10, and 12 above, and further in view of **Kim et al.** (US patent 7,279,568, issued on 10/09/2007, filed on 05/06/2003), is ***withdrawn*** because the claims have been amended.

Amended claim 1 filed on 03/11/2010 reads as follows: An expression system for use in a producer cell line, containing one or more nucleic acid(s) comprising a) at least one nucleic acid for an IL-15/Fc fusion protein, b) at least one promotor and c) at least one nucleic acid for a CD5 leader, the promotor and the nucleic acid for the CD5 leader being functionally linked to the nucleic acid for the IL-15/Fc fusion protein.

None of Ferrari-Lacraz et al., Sutherland et al., and Kim et al. explicitly disclosed the amended limitation "for use in a producer cell line".

The following 103 rejections are necessitated by claim amendments filed 03/11/2010.

5. Claims 1, 2, 4-6, 10, and 12 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Ferrari-Lacraz et al.** (Ferrari-Lacraz et al. An antagonist IL-15/Fc protein prevents costimulation blockade-resistant rejection, *J. Immunol.* 167(6):3478-85, 2001) in view of **Sutherland et al.** (Sutherland et al., Protective effect of CTLA4Ig secreted by transgenic fetal pancreas allografts, *Transplantation*, 69(9):1806-12, 2000; This reference is cited in the IDS filed on 11/13/2006) and **Steurer et al.** (Steurer et al., Ex vivo coating of islet cell allografts with murine CTLA4/Fc promotes graft tolerance. *J. Immunol.* 155(3):1165-74, 1995). *This rejection is necessitated by claim amendments filed 03/11/2010.*

Amended claim 1 filed on 03/11/2010 reads as follows: An expression system for use in a producer cell line, containing one or more nucleic acid(s) comprising a) at least one nucleic acid for an IL-15/Fc fusion protein, b) at least one promotor and c) at least one nucleic acid for a CD5 leader, the promotor and the nucleic acid for the CD5 leader being functionally linked to the nucleic acid for the IL-15/Fc fusion protein.

Ferrari-Lacraz et al. teaches an IL-15 mutant/Fcγ2a protein, a potentially cytolytic protein that is also a high-affinity receptor site specific antagonist for the IL-15Rα receptor protein, as a therapeutic agent. The IL-15-related fusion protein was used as monotherapy or in combination with CTLA4/Fc in murine islet allograft models. As monotherapies, CTLA4/Fc and an IL-15 mutant/Fcγ2a were comparably effective in a semiallogeneic model system, and combined treatment with IL-15 mutant/Fcγ2a plus CTLA4/Fc produced universal permanent engraftment (See abstract, Ferrari-Lacraz et al., 2001).

With regard to the limitation "Fc part of the fusion protein is an Fc fragment of an immunoglobin G" recited in claim 4 of instant application, Ferrari-Lacraz et al. teaches that an IgG2a protein bearing the same Fc sequences as CTLA4/Fc and IL-15 mutant/Fcγ2a protein was used as a control (See left column, page 3479, Ferrari-Lacraz et al.)

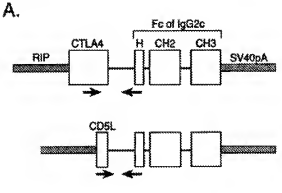
Ferrari-Lacraz et al. does not explicitly teach **(i)** an expression system for use in a producer cell line and a promoter expressing the IL-15/Fc fusion protein recited in claim 1, CMV promoter recited in claim 2, a selectable marker gene recite din claim 5, and **(ii)** the limitation CD5 leader being functionally linked to the nucleic acid for the IL- 15/Fc fusion protein recited in claim 1, and the polyadenylation signal recited in claim 6.

(i) With regard to the limitation "for the use in a producer cell line" recited in claim 1, **Steurer et al.** (1995) teaches mCTLA4/Fc expression and purification: To achieve stable expression, 20 pg of the murine CTLA4Fc plasmid construct was linearized by *PvuI* digestion (New England Biolabs) and electroporated into 10^7 CHO-Kl cells. Transformed CHO-K1 cells were selected with 1 mg/ml G418 (Life Technologies, Inc., which reads on selection of kanamycin resistant gene expression encompassed by the limitation of claim 5) and subsequently

cloned by limiting dilution. Steurer et al. (1995) teaches that established cell lines (i.e. CHO-K1 cell lines for production of CTLA4Fc fusion protein) were then screened for mCTLA4Fc production by an ELISA specific for murine IgG2a. High producing clones were cultured in serum-free media for 12 days. Steurer et al. further teaches CMV promoter (which reads on the limitation of claim 2) for expression of CTLA4Fc fusion protein (See .right column, page 1166, Steurer et al., 1995).

It is noted that Steurer et al. (1995) is the reference cited by Ferrari-Lacraz et al. (See treatment protocol, left column, page 3479, Ferrari-Lacraz et al., 2001) under Materials and Methods regarding treatment protocol and expression of Fc fusion protein *ex vivo* from producer cell lines.

(ii) With regard to the limitation CD5 leader being functionally linked to the nucleic acid for the IL- 15/Fc fusion protein recited in claim 1, and the polyadenylation signal recited in claim 6, **Sutherland et al.** teaches cDNA encoding the murine CTLA4 was fused to IgG2c Fc (CTLA4Ig) and CD5 leader sequence was fused to the Fc of mouse IgG2c (a positive control, CD5LIg, for expression of a Fc fusion protein) with SV40 polyadenylation signal (SV40pA), and expressed transgenically under the control of the rat insulin promoter (RIP) in C57BL/6 mice (See Materials and methods, Fig. 3A, shown below, Sutherland et al., 2000).



Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time of the invention to combine the teachings of Ferrari-Lacraz et al. regarding as monotherapies, CTLA4/Fc and an IL-15 mutant/Fcγ2a fusion proteins are comparably effective in a semiallogeneic model system, with the teachings of (i) Steurer et al. regarding CHO-K1 producer cell lines for production of CTLA4/Fc fusion protein, and the teachings of (ii) Sutherland et al. regarding cDNA encoding the murine CTLA4 was fused to IgG2c Fc (CTLA4Ig) and CD5 leader sequence was fused to the Fc of mouse IgG2c (i.e. a positive control, CD5LIg, for protein expression), and expressed transgenically under the control of the rat insulin promoter (RIP) for allografts, whereas production of CTLA4Fc fusion protein from producer cell line CHO-K1 can be used for promoting graft tolerance, to arrive at the claimed expression system recited in claims 1, 2, 4-6, 10, and 12 of instant application, by linking nucleic acid encoding CD15 leader sequence taught by Sutherland et al. to the nucleic acid encoding IL-15/Fc fusion protein taught by Ferrari-Lacraz et al. under a CMV promoter and express said nucleic acid in CHO-K1 producer cell line taught by Steurer et al.

One having ordinary skill in the art would have been motivated to combine the teachings of Ferrari-Lacraz et al. with the teachings of Steurer et al. and the teachings of Sutherland et al. because (i) Ferrari-Lacraz et al. cites the work by Steuer et al. under "Materials and Methods" for details of construction and expression of CTLA4Fc fusion protein, whereas (ii) Steurer et al. specifically teaches *ex vivo* production of CTLA4Fc fusion protein driven by a CMV promoter in CHO-K1 producer cell line and the use of CTLA4Fc fusion protein to promote graft tolerance, and (iii) Sutherland et al. teaches CD5 leader sequences as an established positive control for

expression of a Fc fusion protein and use of a promoter (RIP, a rat insulin promoter) to direct the expression of CTLA4/IgG2c Fc fusion protein.

There would have been a reasonable expectation of success given (i) successful demonstration of CTLA4/Fc and an IL-15 mutant/Fcγ2a fusion proteins are comparably effective in a semiallogeneic model system, by the teachings of Ferrari-Lacraz et al., (ii) successfully demonstration of *ex vivo* production of CTLA4Fc fusion protein driven by a CMV promoter in CHO-K1 producer cell line and the use CTLA4Fc fusion protein to promote graft tolerance, by the teachings of Steurer et al., and (iii) successfully demonstration of cDNA encoding the murine CTLA4 was fused to IgG2c Fc (CTLA4Ig) and CD5 leader sequence was fused to the Fc of mouse IgG2c (i.e. a positive control, CD5LIg, for expression of Fc protein), and expressed transgenically under the control of the rat insulin promoter (RIP) for allografts, by the teachings of Sutherland et al. It is noted that the level of skill in art of molecular cloning is high. Absent evidence from the contrary, one of ordinary skill in the art would have reasonable expectation of success to link nucleic acid encoding CD5 leader taught by Sutherland to the nucleic acid encoding IL-15/Fc fusion protein taught by Ferrari-Lacarz et al. under a CMV promoter and express said nucleic acid in a producer cell line (i.e. CHO-K1) taught by Steurer et al.

Thus, the claimed invention as a whole was clearly *prima facie* obvious.

6. Claims 3 and 21 are rejected under 35 U.S.C. 103(a) as being unpatentable over **Ferrari-Lacraz et al.** (Ferrari-Lacraz et al. An antagonist IL-15/Fc protein prevents costimulation blockade-resistant rejection, *J. Immunol.* 167(6):3478-85, 2001) in view of **Sutherland et al.** (Sutherland et al., Protective effect of CTLA4Ig secreted by transgenic fetal pancreas allografts,

Transplantation, 69(9):1806-12, 2000; this reference is cited in the IDS filed on 11/13/2006) and **Steurer et al.** (Steurer et al., Ex vivo coating of islet cell allografts with murine CTLA4/Fc promotes graft tolerance. *J. Immunol.* 155(3):1165-74, 1995), as applied to claims 1, 2, 4-6, 10, and 12 above, and further in view of **Kim et al.** (US patent 7,279,568, issued on 10/09/2007, filed on 05/06/2003). *This rejection is necessitated by claim amendments filed 03/11/2010.*

The teachings of Ferrari-Lacraz et al. (2001), Sutherland et al. (2000), and Steurer et al. (1995) have been discussed in the preceding section of the rejection of claims 1, 2, 4-6, 10, and 12 under 35 U.S.C. 103(a) as being unpatentable over Ferrari-Lacraz et al. in view of Sutherland et al. and Steurer et al.

None of Ferrari-Lacraz et al. (2001), Sutherland et al. (2000), and Steurer et al. (1995) explicitly teaches a CMV promoter with intron A being part of a transcription-regulating unit as recited in claims 3 and 21.

However, at the time of filing of instant application, a CMV promoter with intron A as part of a transcription-regulating unit was known in the art. For instant, **Kim et al.** teaches a chimeric expression vector having the advantages of both pCN and pEF vectors. Particularly, the chimeric expression vector pCEF of the present invention is constructed by inserting the enhancer region of HCMV IE gene into upstream of the EF1α gene promoter of pEF vector while maintaining the EF1α gene-derived transcription regulatory element comprising the promoter, the entire exon 1 sequence, the entire intron A sequence and the nucleotide sequence just before the initiation codon ATG of exon 2, which results in a high level of gene expression both in transient and stable expression systems (See lines 9-20, column 4, and Example 1, Kim et

al.). Kim et al. further teaches Neomycin resistant gene as a selectable marker (See for instance, Fig. 13 and Example 1, Kim et al.)

Therefore, it would have been *prima facie* obvious to one having ordinary skill in the art at the time of the invention to incorporate the teachings of Kim et al. regarding CMV promoter with intron A as a transcription-regulating unit, into the combined teachings of Ferrari-Lacraz et al. (2001), Steurer et al. (1995), and Sutherland et al. (2000) directing to an expression system for use in a producer cell line, containing one or more nucleic acid(s) comprising a) at least one nucleic acid for an IL- 15/Fc fusion protein, b) at least one promoter and c) at least one nucleic acid for a CD5 leader, the promoter and the nucleic acid for the CD5 leader being functionally linked to the nucleic acid for the IL- 15/Fc fusion protein, to arrive at the claimed expression system recited in claims 3 and 21.

One having ordinary skill in the art would have been motivated to incorporate the teachings of Kim et al. into the combined teachings of Ferrari-Lacraz et al. (2001), Steurer et al. (1995), and Sutherland et al. (2000) because Kim et al. teaches that gene expression controlled by CMV promoter with intron A as a transcription-regulating unit leads to a high level of gene expression both in transient and stable expression systems.

There would have been a reasonable expectation of success given **(i)** successful establishment of an expression system for use in a producer cell line, containing one or more nucleic acid(s) comprising a) at least one nucleic acid for an IL- 15/Fc fusion protein, b) at least one promoter and c) at least one nucleic acid for a CD5 leader, the promoter and the nucleic acid for the CD5 leader being functionally linked to the nucleic acid for the IL-15/Fc fusion protein, by the combined teachings of Ferrari-Lacraz et al. (2001), Steurer et al. (1995), and Sutherland et

al. (2000), and **(ii)** the construction of expression vector having gene of interest expressed by a CMV promoter with intron A as a transcription-regulating unit would lead to a high level of gene expression both in transient and stable expression systems, by the teachings of Kim et al.

Thus, the claimed invention as a whole was clearly *prima facie* obvious.

Applicant's arguments and Response to Applicant's arguments

Applicant's remarks filed on 03/11/2010 regarding the previous rejection of record are addressed as the related to the new grounds of rejection set forth above. It is noted that Steurer et al. (1995) has been recited to address the amended limitation "an expression system for use in a producer cell line" recited in claim 1.

Applicant argues that the Office has used hindsight in combining the teachings of Ferrari-Lacraz and Sutherland to come to the claimed invention. The Office must identify a motivation to combine the references that create the case of obviousness. In re Rouffet, 149 F.3d 1350, 1357, 47 U.S.P.Q.2d 1453, 1457 (Fed. Cir. 1998). That is, "the examiner must show reasons that the skilled artisan, confronted with the same problems as the inventor and with no knowledge of the claimed invention, would select the elements from the cited prior art references for combination in the manner claimed." (Emphasis added.) The Office, in this instance, has not met this requirement. Even if the Office identifies every element of a claimed invention in the prior art, this alone is insufficient to negate patentability. Otherwise, "rejecting patents solely by finding prior art corollaries for the claimed elements would permit an examiner to use the claimed invention as a blueprint for piecing together elements in the prior art to defeat the patentability of the claimed invention. Applicant states that the law requires more than conclusory statements, it requires evidence, and the Office has provided none (See Applicant remarks pages 7-8 filed on 03/11/2010).

In response, The Examiner would like to direct Applicant's attention to recent decision by U.S. Supreme Court in *KSR International Co. v. Teleflex, Inc.* that forecloses the argument that a **specific** teaching, suggestion, or motivation is an absolute requirement to support a finding of

obviousness. See recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing KSR, 82 USPQ2d at 1936) (available at http://www.uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf). The Examiner notes that in the instant case, even in the absence of recent decision by U.S. Supreme Court in *KSR International Co. v. Teleflex, Inc.,* the suggestion and motivation to combine Ferrari-Lacraz et al., Sutherland et al., and Steurer et al. have been clearly set forth above in this office action.

As stated in the new rejections in this office action, Sutherland et al. teaches cDNA encoding the murine CTLA4 was fused to IgG2c Fc (CTLA4Ig) and CD5 leader sequence was fused to the Fc of mouse IgG2c (a positive control, CD5LIg, for expression of a Fc fusion protein) with SV40 polyadenylation signal (SV40pA), and expressed transgenically under the control of the rat insulin promoter (RIP) in C57BL/6 mice (See Materials and methods, Fig. 3A, shown below, Sutherland et al., 2000).

Furthermore, to arrive at the claimed expression system recited in claims 1, 2, 4-6, 10, and 12 of instant application, by linking nucleic acid encoding CD15 leader sequence taught by Sutherland et al. to the nucleic acid encoding IL-15/Fc fusion protein taught by Ferrari-Lacraz et al. under a CMV promoter and express said nucleic acid in CHO-K1 producer cell line taught by Steurer et al. In this regard, the level of skill in art of molecular cloning is high. Absent evidence from the contrary, one of ordinary skill in the art would have reasonable expectation of success to link nucleic acid encoding CD5 leader taught by Sutherland to the nucleic acid encoding IL-15/Fc fusion protein taught by Ferrari-Lacarz et al. under a CMV promoter and express said nucleic acid in CHO-K1 producer cell line taught by Steurer et al.

It is worth noting that the combined teachings of Ferrari-Lacraz et al., Sutherland et al., and Steurer et al. encompasses both *in vivo* (Ferrari-Lacraz et al., Sutherland et al.), and *in vitro* (Steurer et al.) aspects of the usign the claimed expression system, which is broader and more advanced than the intended *in vitro* use (i.e. the amended limitation "for use in a producer cell line" recited in claim 1) claimed by the Applicant. Therefore, Applicant's arguments that the 103 rejections of record rely on "hindsight" are not persuasive because the specification of instant application does not provide any example pertaining to the *in vivo* use of the claimed expression system. In fact, the specification of instant application does not even contemplate *in*

vivo use of the claimed expression system. To the contrary, the combined teachings of cited prior arts disclose clear scientific evidences of successful performance (i.e. reduce to practice) of *in vivo* (as well as *in vitro*) use of the claimed expression system in the context of overcoming allograft rejection in transgenic mice (as well in CHO-K1 producer cell line).

Finally, it is noted that one cannot show nonobviousness by attacking references individually where the rejections are based on combinations of references. See *In re Keller*, 642 F.2d 413, 208 USPQ 871 (CCPA 1981); *In re Merck & Co.,* 800 F.2d 1091, 231 USPQ 375 (Fed. Cir. 1986).

Conclusion

7. No claim is allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Applicant is reminded that upon the cancellation of claims to a non-elected invention, the inventorship must be amended in compliance with 37 CFR 1.48(b) if one or more of the

currently named inventors is no longer an inventor of at least one claim remaining in the application. Any amendment of inventorship must be accompanied by a request under 37 CFR 1.48(b) and by the fee required under 37 CFR 1.17(i).

Any inquiry concerning this communication from the examiner should be directed to Wu-Cheng Winston Shen whose telephone number is (571) 272-3157 and Fax number is 571-273-3157. The examiner can normally be reached on Monday through Friday from 8:00 AM to 4:30 PM. If attempts to reach the examiner by telephone are unsuccessful, the supervisory patent examiner, Peter Paras, Jr. can be reached on (571) 272-4517. The fax number for TC 1600 is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Wu-Cheng Winston Shen/
Primary Examiner
Art Unit 1632